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Atrophic Rhinitis in Pigs other than Landrace

BY
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History

IN February, 1956, a herd of pigs in the Isle of Man, consisting of two Landrace adults, a litter of six Landrace pigs approximately eight weeks old, and approximately 50 Large White pigs of various ages, was compulsorily slaughtered under the provisions of the Isle of Man Diseases of Animals (Prevention) Acts. Examinations of cross sections of the snouts of all the animals slaughtered were carried out, and it was found that lesions of atrophic rhinitis were present in some of the Landrace pigs and also in some of the Large White pigs.

In view of the owner's statement that there had been no direct contact between some of these Large White pigs and the Landrace it was decided to carry out a survey in the form of an examination of the snouts of all pigs slaughtered in the Isle of Man irrespective of breed or ownership.

FIG. 1.—Section of the snout of a normal Manx Large White pig with a dead-weight of approximately 200 lb.

Methods

The slaughtering of all Manx pigs is carried out in only two public abattoirs. An Order was made by the Isle of Man Board of Agriculture and Fisheries under the Isle of Man Diseases of Animals (Prevention) Acts, requiring that the snouts of all pigs slaughtered in these abattoirs should be removed, identified, and made available to the veterinary officers of the Animal Health Department of the Board for examination.

Identification was relatively simple, as each producer had his pigs "slap marked" before removal to the abattoirs for slaughter and, after the removal of the snout, the identification mark was transferred to the snout in indelible pencil. The snout was removed by a transverse saw cut immediately behind the first premolar tooth.

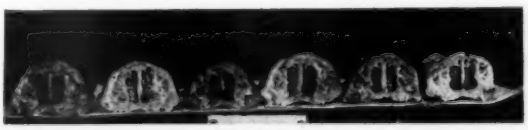

FIG. 2.—Sections of snouts from Landrace pigs from a herd affected with atrophic rhinitis. *N.B.*—Bent nasal septa in the two centre snouts. (Approx. 200 lb. dead-weight.)

Snouts showing abnormality were removed from the abattoir and a large number forwarded to Mr. J. T. Done, at the Ministry of Agriculture veterinary laboratory, Weybridge. In addition to those of Manx pigs, the snouts from some 80 pig carcases which had been imported by air from Northern Ireland were also removed and submitted for examination. When lesions of atrophic rhinitis were found visits were made to the premises of the producer concerned to examine the herd.

Results

During the period of the survey the snouts of approximately 1,000 Manx pigs were examined. Approximately 10 per cent. of those examined showed varying degrees of abnormality (Figs. 3 and 4).

The lesions observed were indistinguishable from those of atrophic rhinitis, but in no instance were they as severe as those found in the worst affected Landrace. Atrophy of the ventral turbinates of either one or both sides was the most common finding. Bending

FIG. 3.—Sections of snouts from Manx Large Whites. *N.B.*—Bent nasal septa in the second and third snouts from left of the picture. (Dead-weights from 160 to 200 lb.)

of the nasal septum was fairly frequently found, but complete atrophy of the dorsal turbinates was rarely found. The bending which occurred in both breeds took place towards the concave side of the septum nasi. In many instances the bending was very slight and insufficient to show clearly in a photograph. In a number of cases it could be observed only after careful examination.

The percentage of abnormal snouts varied from herd to herd and even from group to group in the same herd. For example, producer H.1. entered eight pigs in one week for bacon, of which six were found to have atrophic rhinitis abnormalities. The following week, out of 16 pigs entered, none showed abnormality.

Of the snouts from pig carcases imported from Northern Ireland, 23, or approximately 26 per cent., showed lesions similar to those of the Manx pigs (Fig. 5).

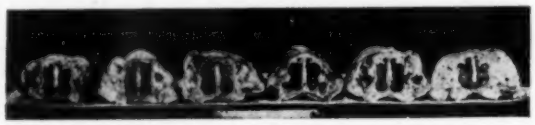

FIG. 4.—Sections of snouts from Manx Large Whites. *N.B.*—Bent nasal septum in the snout on the right of the picture. (Dead-weights 160 to 200 lb.)

A number of normal snouts, both of Landrace and Large White pigs, were boiled to remove the soft tissues. It was noticeable that the quality and strength of bone in the snout of the Landrace was much less than that of Large Whites of similar age and bodyweight. It was found that in certain Large White pigs where atrophy of the turbinates had taken place and where there was bending of the septum nasi, there was also lateral deviation of the bones of the snout which was not observable until the soft tissues had been removed (Figs. 6, 7, and 8). This bending of the bony structure of the snout was similar to that found in two clinical cases of atrophic rhinitis in Landrace pigs (Fig. 6).

Visits to herds producing abnormal snouts revealed that while no animals could be found showing lateral deviation of the snout, it was usual to find some animals showing unilateral or bilateral ocular discharge, and also a number showing a broadening of the snout in the region of the first premolar teeth. This broadening of the snout was found to be a common feature in animals showing abnormalities on section of the snout. The majority of the animals concerned had had no contact direct or indirect with Landrace pigs.

FIG. 5.—Sections of snouts from carcases of pigs imported from N. Ireland. *N.B.*—Bent nasal septa in the two snouts on the right of the picture. (Dead-weights approx. 120 lb.)

Coughing suggestive of virus pneumonia (V.P.P.) was present in most of the herds examined.

Examinations of affected non-Landrace pigs showed that the broadening of the snout was not due to one factor alone. There appeared to be, in many instances, a hypertrophy of the soft structures, particularly muscle. There was also frequently present a bony protuberance in the region of the canine and first premolar teeth but this was not a constant factor.

In the more severely affected cases the bony lateral walls of the snout had lost the normal concavity and had become straight or slightly convex when viewed from the outside.

Discussion

A condition indistinguishable from atrophic rhinitis has been found in Manx pigs and in pigs imported from Ireland. The vast majority of the animals concerned had no contact, direct or indirect, with imported or home-bred Swedish Landrace pigs. The presence of this condition had, until comparatively recently, been unsuspected in British pigs.

William Youatt, whose book, *The Pig*, was published in London in 1847, was evidently familiar with such a disease. In his chapter on Nasal Catarrh he wrote:—

> "It is characterised by defluxion from the nose in the first place and its advance is so gradual as to be almost imperceptible. But it gains ground daily—attacks the respiratory passages—cough and sneezing come on—there is evident difficulty of swallowing, and the respiration is impeded by the mucus formed. After some time the membrane of the nose becomes thickened, the nostril swelled and deformed, and the *snout drawn to one side.*"

While twisting of the snout has not as yet been observed in Manx pigs other than Landrace, there does not appear to be any reason to believe that twisting would not be apparent in the live animal if

the native breeds had the length and narrowness of snout and fineness of bone which are outstanding features of the Landrace.

Twisting of the bony structure in non-Landrace pigs has been found; and while, on the whole, the twisting is somewhat less than in those specimens obtained from Landrace pigs, this could be accounted for by the greater strength and weight of bone in the native pig. While twisting of the snout has been seen in Manx-bred Landrace pigs, it does not appear to be such an outstanding feature in the disease as one is frequently led to believe.

Out of approximately 900 Manx Landrace pigs which have been examined during the last two years, twisting of the snout has occurred in fewer than 1 per cent. of them.

FIG. 6.—Bone structure from the snouts of pigs which had bent nasal septa. Reading from left to right:—Irish Large White, Manx Large White, Landrace, Landrace. In the case of the two Landrace lateral deviation of the snouts was apparent before slaughter.

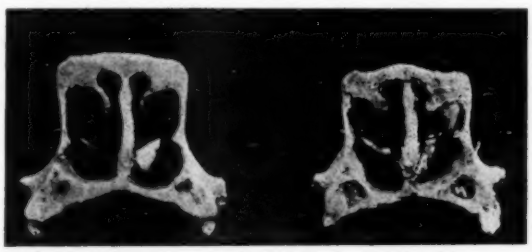

FIG. 7.—Bone structure from second and third snouts from the left of picture Fig. 3. All soft structures have been removed except nasal septa.

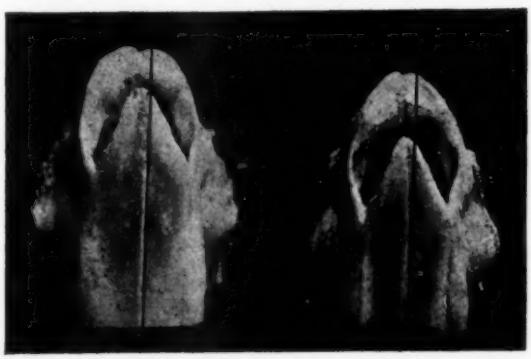

FIG. 8.—The same snouts (as Fig. 7) showing slight bending to the left.

The percentage of abnormal snouts found on *post-mortem* examination of non-Landrace pigs is much the same as that found in Landrace herds where compulsory slaughter has been carried out because of the presence of atrophic rhinitis in the herd.

The abnormalities found have been identical in both Landrace and non-Landrace breeds of pig, with the exception that in non-Landrace the degree of abnormality has never been as marked as that of the most severely affected Landrace pigs.

The *post-mortem* findings have been those described by Swahn of the State Institute of Veterinary Medicine, Stockholm.

The following is a translation from the original French text:—

"This atrophy can vary between a relatively slight shortening of the turbinates and an almost complete atrophy. These modifications generally begin in the centre of the free side of the bony base of the 'ventral' turbinate. As the atrophy progresses, the curled turbinate straightens. The mucosa itself follows the movement, but more slowly. The turbinates, or parts of them, may in serious cases almost disappear. So, in fairly advanced cases, one can see that no *ventral* turbinates remain except for a fold of mucous membrane of about one centimetre and which hangs like a curtain floating in the remainder of the nasal cavity. It is important, in a careful examination, to inspect the whole of the turbinates.

"As well as the modifications of the turbinates in this 'sneezing disease' one can usually see in an anatomo-pathological examination, modifications of the position of the nasal septum. There also, the degree varies considerably, from a slight warping of the 'septum' due to the *ventral* part deviating slightly to one side, to a marked deviation in the form of an arc. It is curious that the deviation is made apparently without exception, not to the side where there is the most marked modification of the turbinates, but to the opposite side. This could be explained by the bony atrophy being more marked in the half of the face where atrophy of the nasal turbinates is stronger so that there results an attraction, to that side, of the dorsal and ventral attachments of the septum.

"When making a large-scale check of snouts, for instance when making a routine examination of hundreds of animals destined for the butcher, and belonging to herds submitted to the regular health control, it is usually sufficient to inspect a section of nasal passages in the region of the canines or just behind them. To facilitate the examination of the shape of the turbinates and the position of the nasal septum, it is very important that the snout is sawn perpendicularly to the longitudinal direction."

In the same report Swahn also states that lateral and bilateral ocular discharges and thickening of the snout in the region of the canines are symptomatic of the disease. He also stresses the difficulties of diagnosis in the live animal except in the case of lateral deviation of the snout. The importance of *post-mortem* examination is also stressed and it can

SYMPOSIUM

Leucosis and Respiratory Disease in Poultry

The first half of this Symposium was published last week.

2- The Structure and Function of the Respiratory Pathways of *Gallus domesticus*

BY

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THE lung of the bird has been described, justifiably, as " the most complicated in the animal kingdom " (Scharnke, 1938). Moreover there appears to be no adequate account of it, for any species of bird, in any of the British, American, and West European text-books of veterinary and comparative anatomy; all contain an undesirable blending of species into a composite but non-existent " bird," as well as radical omissions and inaccuracies. The task of describing briefly the respiratory tract of *G. domesticus* is therefore formidable, and indeed not without danger since some simplifications are inevitable. For all details it is essential to consult Locy & Larsell's (1916) description of the embryology and Campana's (1875) monograph on the definitive anatomy. These works form the basis of the following section, but I have confirmed many of their findings (as far as they relate to the definitive anatomy) by my own observations.

Structure

The primary bronchus. The respiratory tract from the nares to the syrinx is simple and can be omitted. The syrinx gives rise to the two primary bronchi, each of which runs sinuously through the lung and is dilated in its middle section (Figs. 1, 2, and 3).

The secondary bronchi. The primary bronchus gives rise to four groups of secondary bronchi: ventromedial, dorsomedial, lateral, and dorsal. The ventromedial group consists of four large tubes springing from the ventromedial aspect of the cranial part of the primary bronchus (I, II, III, and IV in Fig. 1). The other three groups arise caudal to the expanded mid-section of the primary bronchus: the dorsomedial group consists of six large tubes arising from its dorsomedial aspect (1 to 6 in Fig. 1); the

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Atrophic Rhinitis in Pigs, etc.—*concluded.*

be inferred that twisting of the snout is an " exception rather than the rule " in herds affected with atrophic rhinitis.

It is particularly important in carrying out a survey of pig snouts in order to establish the presence of the condition under discussion, that the animals selected should represent the majority, at least, of the pigs in the area.

It was notable, in the Isle of Man, that a sporadic examination of pig snouts carried out prior to the survey had failed to reveal the presence of any abnormalities, and during actual survey there were periods during which there were few or no abnormalities present.

The finding of a condition indistinguishable on *post-mortem* examination from the less severe types of atrophic rhinitis in non-Landrace pigs, having the symptoms in the live animal of a mild type of atrophic rhinitis, and having approximately the same percentage of abnormal snouts on *post-mortem* examination as Landrace herds known to be affected with atrophic rhinitis, has led the writer to the conclusion that there is no longer any justification for a continuance of a slaughter policy. This view holds good whether the affected herds are Landrace or any other breed of pig.

It would appear that the classic symptom of atrophic rhinitis, lateral deviation of the snout, may, in this country, be confined to the Landrace breed and is nothing more than a manifestation in a susceptible breed of a condition which has existed, in the Isle of Man at least, for many years.

The inference that the disease also exists in other parts of Great Britain cannot be avoided, as for many years store pigs have been exported from the Isle of Man. It is considered that a similar survey, carried out in other parts of Great Britain, would have similar results.

Summary

1. The snouts of 1,000 pigs slaughtered in the Isle of Man were examined.

2. A condition indistinguishable from atrophic rhinitis was found in approximately 10 per cent. of all snouts examined.

3. Examination of the herds concerned showed the presence of some of the clinical signs of atrophic rhinitis. Lateral deviation of the snout in the live animal was not observed.

4. Lateral deviation of the bones of the snout was noted in pigs of both Irish and Manx origin.

Acknowledgment.—The writer is indebted to Mr. J. T. Done, of Weybridge, who carried out examinations of many of the snouts obtained during the survey. His help and advice have been invaluable.

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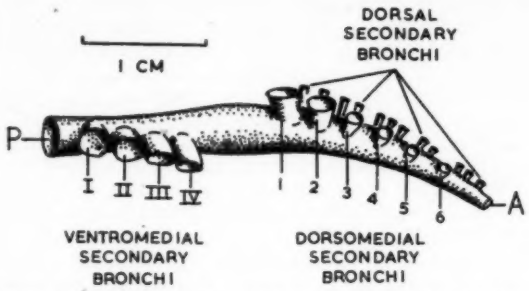

FIG. 1.—Medial view of the right primary bronchus to show its shape and three of its four groups of secondary bronchi. The fourth group, the six lateral secondary bronchi, are invisible from this view. P = the primary bronchus where it enters the lung. A = the caudal end of the primary bronchus, just before it expands into the abdominal air sac.

lateral group consists of six medium-sized lateral tubes; the dorsal group consists of about 20 small dorsal tubes (Fig. 1).

The tertiary bronchi (or parabronchi). After a course of only a few millimetres every secondary bronchus divides into a series of smaller tubes, the tertiary bronchi. Those arising from the four ventromedial secondary bronchi anastomose end to end with those from the six dorsomedial secondary bronchi, forming a series of hoops (Fig. 2): they constitute one functional unit, which makes up about two-thirds to three-quarters of the lung. The tertiary bronchi of the lateral and dorsal groups radiate straight

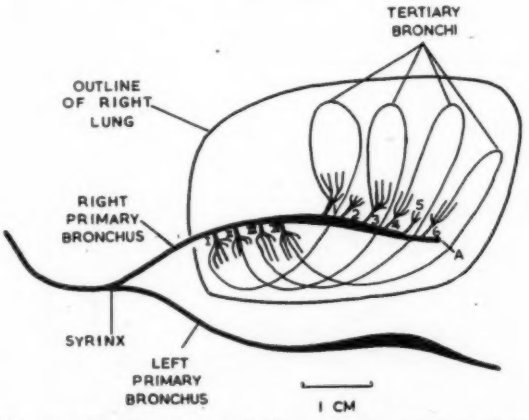

FIG. 2.—Diagram of the right lung showing representatives of the hoop-like tertiary bronchi connecting the ventromedial secondary bronchi (I to IV) to the dorsomedial (1 to 6). For A see Fig. 1. The view is medial, and slightly dorsal.

towards the surface of the lung and are not arranged into organised circuits; they do anastomose with each other and with the system of hoops just described. The approximate proportions of the lung made up by the tertiary bronchi of the four groups of secondary bronchi are shown by Fig. 3.

The air capillaries. These are narrow tubules arising profusely along every tertiary bronchus (Fig. 4). They branch and anastomose freely. The spaces between them are filled with blood vessels, and all authorities agree that the gaseous exchanges occur here.

The main connexions between the lung and the air sacs. The air sacs are an integral part of the lung. Developmentally, each arises as a dilatation of a branch of a secondary bronchus. In this way the first ventromedial secondary bronchus gives rise to the cervical air sac and part of the interclavicular sac; the third ventromedial secondary bronchus forms the rest of the interclavicular sac and all of the cranial thoracic sac. (See Fig. 5.) The third lateral

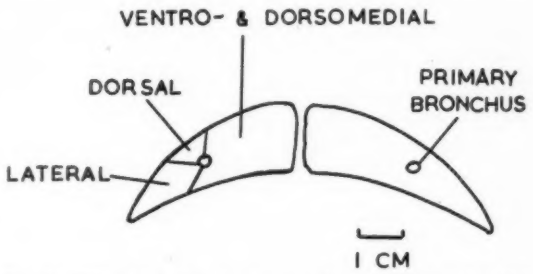

FIG. 3.—Diagrammatic transverse section of the lungs showing the approximate areas occupied by the tertiary bronchi of the lateral, dorsal, and ventro- and dorsomedial secondary bronchi.

secondary bronchus forms the caudal thoracic sac. The abdominal sac is often regarded as a direct continuation of the primary bronchus but is more likely to be a seventh lateral bronchus.

The recurrent bronchi. Each sac except the cervical connects with the lung by additional tubes called the recurrent bronchi (Fig. 5). There are about five for each sac. They anastomose freely with the tertiary bronchi.

The air sacs. The abdominal, caudal thoracic, and cranial thoracic sacs are paired; the interclavicular sac is a mid-line structure. The cervical sac is generally supposed to be paired (*e.g.* Grau, 1943) but is really median.

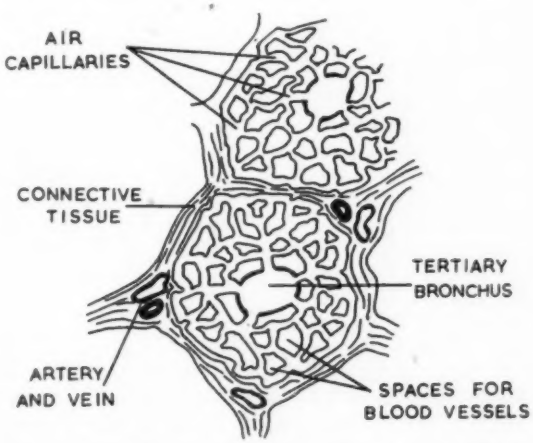

FIG. 4.—Semi-diagrammatic transverse section through a tertiary bronchus to show the air capillaries (magnified).

The relatively large and mobile abdominal sac lies among the caudal viscera. The caudal and cranial thoracic sacs are relatively fixed in shape and position, and lie dorsally in the coelom; the cranial thoracic sac more or less covers the ventromedial surface of the lung. The large and very complicated interclavicular sac occupies the thoracic inlet. It consists of numerous intra- and extra-coelomic diverticula; the former spread between the heart and its neighbouring viscera, and the latter penetrate between the bones and muscles of the pectoral girdle. The cervical sac (Fig. 5) consists of a median chamber which leads

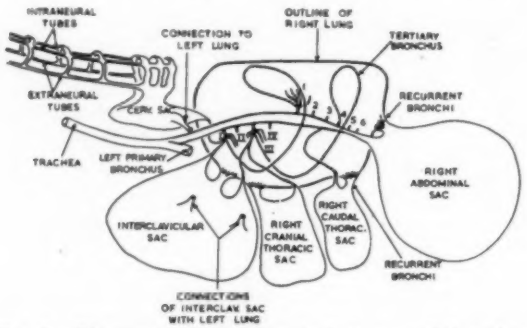

FIG. 5.—Diagram of the right lung and air sacs. Orientation as for Fig. 2. The secondary bronchi, two tertiary bronchi, and the main connexions to each sac, are each represented by only a single line. The caudal thoracic sac connects to the primary bronchus by a branch of the third lateral secondary bronchus. The sizes of the sacs are not in proportion either to each other or to the lung. The numerals I to IV and 1 to 6 mean the same as in Figs. 1 and 2. Cerv. sac. = cervical air sac.

on each side to a pair of tubes, one inside the neural canal (intraneural) and the other outside (extraneural); the latter goes through the foramen transversarium as far as the axis.

The aerated bones. The cervical, interclavicular, and abdominal sacs invade the skeleton. Many opinions exist about which bones are affected, but the problem has recently been clarified (King, submitted for publication). The cervical sac aerates the vertebral column from the third cervical to the fifth thoracic vertebra inclusive, and the first two vertebral ribs. The interclavicular sac aerates the humerus, coracoid, and sternum. The abdominal sac aerates the pelvic bone and the remaining vertebrae except the free coccygeal and pygostyle.

Function

Space allows only a brief survey of the mass of experimental and theoretical evidence on the movement of air through the lungs and air sacs during breathing. There are two great problems: how is the air moved in and out, and what route does it take in the lungs?

The movement of air. There are two theories. The bellows theory, suggested by Perrault some 20 years after Harvey (1651) described the air sacs, claims that the lungs remain virtually constant in size during the breathing cycle, while the sacs by alternate dilatation and reduction suck and blow the air through the lungs. This is an obvious deduction from the anatomy. It is also supported by experimental evidence: similar changes in pressure occur synchronously in all the sacs during the breathing cycle (Francois-Franck, 1906), and breathing soot-laden air results in heavy deposition of the particles on to the walls of the sacs (Dotterweich, 1930).

The other theory proposes that the sacs are inactive but the lung itself is alternately expanded and contracted by the movement of the ribs, thus drawing in and expelling the air (Victorow, 1909). Soum (1896) actually recorded these lung movements, and also showed with Victorow (1909) that the pigeon at any rate can survive when all the sacs are opened to the outside air or plugged, which is certainly hard to explain by the bellows theory.

The dispute continues, for Makowski (1938) and Scharnke (1938) still believed in the breathing movements of the lung, while Zeuthen (1943) and Hazelhoff (1943) preferred the bellows theory.

The route taken by the air in the lungs. Once again there are two theories. Brandes (1924) initiated the idea that there might be a *circulation* of air in the lung tubes. The proof that there are no valves to control such a circulation (Scharnke, 1938) shook this theory, but the postulation of control by aerodynamic factors (Dotterweich, 1936; Hazelhoff, 1943) has strongly revived it. The best experimental evidence for it comes from studying the site where soot is deposited in the lung after breathing soot-laden air; the result (Fig. 6) strongly suggests that much of the air both in inspiration and expiration must revolve from the dorsomedial secondary bronchi, through the tertiary bronchi, into the ventromedial secondary bronchi. Hazelhoff has called this the d-p-v circuit (d = dorsomedial secondary bronchi, p = tertiary bronchi, and v = ventromedial secondary bronchi), and has shown on glass models that aerodynamic factors could cause it. Our experimental work at Bristol (Biggs & King, 1955) supports this sort of circulation.

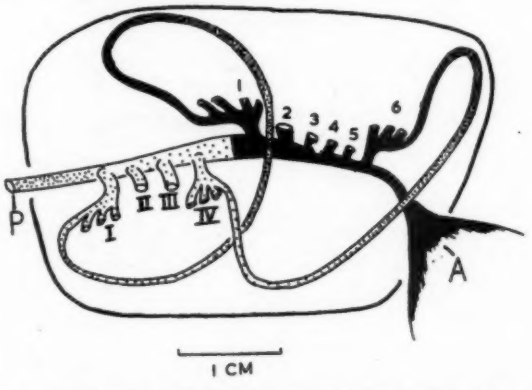

FIG. 6.—Diagram of the right lung, with the same symbols and orientation as Figs. 1 and 2, to illustrate the approximate sites where soot is deposited after breathing sooty air (as described by Hazelhoff, 1943). The heaviest deposits are shaded black; the lighter shaded areas indicate relatively smaller deposits.

Convincing though the circulatory theory may be it is still strongly opposed. The rival theory holds that the air merely oscillates reciprocally in and out of all the lung tubes in inspiration and expiration. This simpler alternative is reached partly through the exposure of theoretical faults inherent in any glass model (Scharnke, 1938), and partly through direct evidence from gas analyses (Zeuthen, 1943).

Anatomy Applied to Pathology

The facts about normal structure and function as a rule are useful as a general basis for the study of abnormality. Occasionally they also help to unravel the pathogenesis of particular diseases. For example, do the lower respiratory lesions of an air-borne infection differ in site from those of a blood-borne infection? I would expect them in the former instance to be concentrated in certain regions of the lung; if the d-p-v circuit really occurs, and there is good evidence that it does, one would expect the lesions to be far more numerous in the dorsomedial segment (the half nearer to the vertebral column), *i.e.* the area occupied by the dorsomedial secondary bronchi and their tertiary bronchi. Again with air-borne infection there is every reason to expect the air sacs to be affected as well as the lungs, especially if the bellows theory is right. On the other hand with blood-borne infection all parts of the lung appear equally vulnerable: the air sacs might be relatively little affected, being far less vascular than the lung. Finally, do the bones become infected in chronic air sac infection? The penetration of the air sacs into the bones suggests osteomyelitis as a theoretical possibility.

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NEW TITLE FOR SANITARY INSPECTORS

By an Act of Parliament dated August 2nd, those previously known as sanitary inspectors will in future be called public health inspectors. The full title of the Act is Sanitary Inspectors (change of short title designation) Act, 1956.

Differential Diagnosis and Control of Respiratory Disease in the Field

BY

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WITH due respect to this morning's speakers on leucosis, I doubt whether even they have succeeded in establishing such a multiplicity of conditions as exists within the group loosely termed "respiratory disease." Whatever the pathological complexities may be of leucosis they do not present the same difficulties to the diagnostician as the differentiation of the numerous diseases affecting the respiratory tract of the fowl. Not only are there at least 10 quite distinct diseases which commonly give rise to respiratory symptoms in poultry, but symptoms are common to several diseases while more than one respiratory disease may co-exist in the same bird or in the same flock. Nevertheless if the fullest advantage is to be taken of recent advances in the control of these diseases by biological products or chemotherapeutics, then some effort must be made to differentiate the conditions with a reasonable degree of accuracy.

Unfortunately many of these diseases can only be differentiated by laborious and time-consuming laboratory techniques, and the time lag resulting often renders the diagnosis more of academic interest than of practical value to the farmer, whose main concern is not to establish a diagnosis but to control his losses. The following quotation from Chu (1954) illustrates the techniques involved; "Newcastle disease is differentiated by the haemagglutination-inhibition test carried out in the sera of infected fowls. Fowl pox and infectious laryngo-tracheitis are diagnosed by isolating the viruses on the chorio-allantoic membrane of 12-day embryonated eggs. Infectious bronchitis is diagnosed by serum neutralisation tests and by isolation of the virus—the growth of the virus on eggs being detected by dwarfing and a lethal effect on the embryos and by the direct demonstration of the virus in the allantoic fluid with the electron microscope. Aspergillosis is diagnosed by microscopic and cultural examinations of infected tissue. Vitamin A deficiency is ruled out by estimation of vitamin A levels in the liver." Certainly in routine laboratory and field advisory work this type of differential diagnosis would be quite impracticable and in most cases one must resort to empirical diagnosis by a process of elimination, while frequently arriving at the method of control by trial and error.

There is undoubtedly a great need for improved facilities in this country and one would like to see the establishment of a unit specialising in the differential diagnosis of respiratory diseases to which material could be sent for identification and confirmation. At the present moment, in view of the widespread incidence of the sub-acute form of Newcastle disease one cannot proceed to the final identification of respiratory infection without first eliminating the presence of that disease. The position might well be summarised by stating that in all

cases where respiratory symptoms occur the outbreak must in the first instance be reported to the Animal Health Division, and that further investigation cannot be undertaken until a negative report has been received. We ourselves have adopted this procedure for the last two or three years and, irrespective of *post-mortem* findings, where there is a history indicative of respiratory or nervous symptoms, particularly in chicks or growers, or of a decrease in egg production in adults, the case is reported. Admittedly, we may have sent our colleagues of the Animal Health Division on many a wild-goose chase but the number of primary outbreaks of Newcastle disease which have been brought to light has in both our opinions more than justified the procedure. It is one which I cannot too strongly recommend to any laboratory handling poultry carcases and similar diagnostic material

There is a voluminous literature on respiratory diseases of the fowl, and with the recent spate of publications in the U.S. on chronic respiratory disease it must now nearly equal that on leucosis. It has been suggested by recent visitors to America that poultry pathologists in that country have welcomed the advent of C.R.D. as a happy release from the frustrations of leucosis research! Under the circumstances the best that can be done in a limited time is to describe briefly each of the commoner diseases in which respiratory symptoms occur, and for your guidance an attempt has been made to summarise in chart form the salient features of these diseases. It must be borne in mind, however, that this chart is little more than a guide and suffers from the usual pitfalls and inaccuracies resulting from over-simplification. For a more detailed description of respiratory disease reference should be made to a paper by Wilson (1954) presented to the FAO Conference in Paris and re-published in THE VETERINARY RECORD; for more recent work, particularly on C.R.D. to the Proceedings of the 80th, 90th, and 91st Annual Meetings of the A.V.M.A. (1952, 1953, and 1954), and to the paper by Chu (1954) in the Proceedings of the 10th World's Poultry Congress. The therapeutic control of respiratory diseases was adequately dealt with by Blount (1955). Newcastle disease and its differential diagnosis has been reviewed by Beaudette (1943, 1950).

For simplicity the diseases will be dealt with systematically under viral, bacterial, fungal, nutritional, and parasitic origin, and not necessarily in their order of importance. Some of the conditions are not primarily respiratory in aetiology and will merely be referred to in passing.

I. VIRAL

(1) Fowl Pox

This disease is far less common than it was pre-war and it generally presents little difficulty in diagnosis, for although the symptoms of the oculo-nasal type are indistinguishable from those of coryza and even Newcastle disease, it is our experience that no outbreak occurs solely in this form. Examination of the remainder of the affected group invariably reveals typical pox lesions affecting the comb, wattles, eyelids and face or characteristic diphtheritic mouth lesions. A diagnosis can readily be established by swabbing the scarified comb of a susceptible cockerel with the suspected material. Typical pox lesions will appear in about four to five days afterwards. Embryonated eggs can also be used for diagnosis. To some extent the diphtheritic mouth lesions can be distinguished from those occurring in coryza in that the latter are readily detached from the underlying mucous membrane, whereas in fowl pox the deposits are firmly adherent and when removed leave a raw, underlying area. The disease tends to spread somewhat slowly in a flock and if detected in the early stages the slaughter of affected birds and careful observation of the remainder with isolation of early cases may halt further spread. The vaccine most commonly used in this country is prepared from pigeon pox virus and applied to the feather follicles of the leg. Immunity, however, only lasts for three to four months and in some active outbreaks it has appeared to be of somewhat limited value. Fowl pox virus vaccine is widely used in the U.S. and Europe and it is generally applied by the stick or wing stab method. It is, however, contra-indicated in the case of birds in production or in chicks where intercurrent disease is present. Although immunity is lasting, there are certain risks attendant on its use.

(2) Infectious Laryngo-tracheitis

During the two or three years following the first report of the disease in this country (Dobson, 1935) numerous outbreaks were encountered, particularly in the fattening plants in the Sussex area. During the war, owing to the disappearance of the table poultry industry, outbreaks were extremely rare. There are now indications that the disease is increasing in incidence and becoming of some importance, particularly in heavily populated areas such as Lancashire. Like fowl pox, the disease, at least in the peracute form, does not present much difficulty in diagnosis and the symptoms and lesions are fairly constantly characteristic. The bird extends the neck fully to make a gasping inhalation through the wide open beak and this is accompanied by rattling, sneezing, and gurgling sounds, the bird shaking its head in an attempt to expel the tracheal obstruction. Blood or blood-clots may be coughed up and can be observed on the dropping boards and walls of the house. In the peracute form the mortality rate may be as high as 50 to 80 per cent., death usually occurring within a day or two of the appearance of symptoms. *Post-mortem* lesions are almost pathognomic and are restricted entirely to the trachea, the upper third or two-thirds of which may be partially or completely occluded with blood casts or blood-tinged fibrinous exudate.

In the chronic or mild type, morbidity may be as low as 2 to 5 per cent. but the mortality rate, although on a flock basis low, is generally 100 per cent. of the affected birds. Symptoms may extend over a period of months with intermittent coughing, gasping respiration, particularly when the bird is excited, and diphtheresis may be seen on the glottis and larynx. In the chronic form, the tracheal casts are generally of semi-solid caseous material. These lesions may be

difficult to differentiate from the diphtheritic form of fowl pox but a definite diagnosis can be established by the inoculation of tracheal suspensions, intratracheally into susceptible fowls, or into 12-day-old embryos. The control of this disease is complicated by the fact that recovered birds invariably remain carriers for periods of well over 12 months so that in many instances not only the slaughter of affected birds and their immediate contacts is necessary, but the complete depopulation of the plant. No vaccine is available in this country but in the United States and Australia vaccination is carried out by the application by means of a stiff brush of egg-propagated virus to the cloacal mucous membrane. The vaccine is reported to be efficient but should be used only under controlled conditions.

In certain parts of Australia infectious laryngotracheitis is a scheduled disease, although the measures applied differ from state to state. In most instances it is obligatory on stock owners to report outbreaks of I.L.T. and owners desirous of vaccinating must apply for a permit. A permit is usually issued only after the owner has received a demonstration on the correct method of vaccination and instruction in the care of handling the vaccine. After vaccination the farm is placed under certain quarantine restrictions. In certain states vaccination must be carried out by a qualified veterinary surgeon and only with approved vaccines.

(3) Newcastle disease

In view of my earlier remarks, this disease requires little comment, since it is obligatory that any veterinary surgeon suspecting the presence of the disease should immediately notify the Animal Health Division. The peracute form, or as it is known the Asiatic type, is fairly readily recognisable both clinically and on autopsy and is unlikely to be mistaken for other respiratory conditions. The mild, or American type, is extremely difficult to diagnose and occurs in a variety of forms and in many instances is subclinical. In young chicks there is gasping and coughing, frequently followed by nervous symptoms such as twisting of the head and neck, and partial or complete paralysis; in growing stock the respiratory symptoms are usually accompanied by nervous complications. In some outbreaks it may be equally difficult to differentiate the condition from such nervous syndromes as encephalomalacia or epidemic tremor. In adult birds there may be slight sneezing and coughing with little obvious nasal discharge; occasionally a green diarrhoea is present but in even the mildest outbreaks there is invariably a sudden drop in egg production accompanied by the laying of soft-shelled, malformed, and even shell-less eggs. Egg production may cease but usually returns to its normal level in from three to six weeks, although shell quality may remain poor. A definite diagnosis of the mild form is rarely possible on clinical grounds. Laboratory diagnosis need not concern us here since it is in the capable hands of the Animal Health Division, but it depends mainly on the use of the haemagglutination-inhibition test, or in certain instances the isolation of virus and in virus serum neutralisation tests. Again, in view of the policy in this country, reference need not be made to the extensive literature on Newcastle disease vaccines and vaccination except to refer to the evidence accumulating on the possible transmission particularly of C.R.D. by contaminated vaccines. The most recent approach, particularly in Canada, has been the use of combined infectious bronchitis/Newcastle disease vaccines applied as sprays (Crawley, 1954).

(4) Infectious bronchitis

Infectious bronchitis was reported in this country by Asplin (1950) and since then we have reason to believe that a number of outbreaks have been encountered. Asplin (1956) considers that the disease may be fairly widespread in Great Britain. Certainly, in the United States and Canada, the disease is of great economic importance and causes serious mortality and impaired growth, particularly in chicks and broilers. In adults although mortality is negligible, the reduction in egg production is severe and costly. The symptoms in chicks are practically indistinguishable from those of Newcastle disease. The incubation period is short (two to three days): there is difficulty in breathing, some coughing, snuffling and sneezing. Mortality may be as high as 60 per cent. and recovered birds appear to be immune. Nervous symptoms have not been reported. In adults the fall in egg production is usually more persistent than in Newcastle disease and the bulk of the affected flock may never return to economic production. The laying of eggs so abnormal as to be unmarketable is a common feature and in one outbreak which we observed in a laying battery, the owner claimed that 2,500 dozen eggs were lost during the season. These eggs showed the most extraordinary shapes and shell texture, some being elongated, others ridged; in many cases rough concretions of calcium appeared on the shell; in some the shells were marble-like and in others areas were missing completely. Definite diagnosis can only be carried out by the demonstration of the virus by serum neutralisation tests, or by the inoculation of embryos in which the dwarfing effect mentioned by Chu is said to occur. There is again an extensive American literature on the subject. In America vaccines are fairly widely used; frequently by the spray technique. Older birds must be vaccinated about a month before going into production and in New York State about 1 per cent. of each flock are inoculated with egg-propagated virus and are introduced into the flock to produce a natural spread of immunity. This method is stated by Levine (1951) to be highly effective.

II. BACTERIAL

(1) Infectious coryza

Until recently this term was used for a number of diseases in which the predominant symptom was an oculo-nasal discharge, and considerable confusion has arisen over the classification of the different types. Although the position is still by no means clear the recent work on C.R.D. would indicate that this latter disease is a quite separate entity from the disease originally described by de Blieck (1931-2) and which

is caused by *Haemophilus gallinarum*. This is probably the mildest form of respiratory disease and to some extent can be compared with the common cold. In our experience it is relatively common in this country, although Chu (1954) in his survey of coryza in England states that out of 35 outbreaks investigated only one was caused by *Haemophilus*. The fact remains that many outbreaks in this country undoubtedly respond to therapy with sulphonamides, drugs against which the C.R.D. agent is stated to be strongly resistant. The incubation period of infectious coryza is usually about one to three days, and generally the disease is of short duration in the flock, frequently clearing up within two to three weeks. Generally symptoms are fairly mild, starting with a thin, watery discharge from the eyes and nostrils. This discharge becomes viscid and as a result of occlusion of the nares exudate accumulates in the infra-orbital and frontal sinuses causing swelling of the face visible from the exterior. In the early stages the facial swellings are oedematous but later as the exudate inspissates they become semi-solid causing gross distortion of the face and even complete disappearance of one or both eyes. Deposits of yellow caseous material occur in the mouth and in the nasal cleft. Mortality as a rule is low and there may be some fall in egg production in adults or impaired growth in younger birds accompanied by coughing and sneezing.

Haemophilus coryza responds fairly quickly to sulphonamides. Sulphathiazole is the most commonly used at a level of 0.125 per cent. in the drinking water. Sulphadimidine at 0.2 per cent. in the drinking water is also effective. Empirically, certain owners have stated that furazolidone at 0.04 per cent. in the food has been effective in the treatment of early cases. At the moment there is no experimental proof of this. Bornstein & Samberg (1953) reported that the intramuscular injection of 0.2 g. streptomycin produced a remarkable clinical recovery and return of egg production in affected fowls.

(2) Chronic respiratory disease (C.R.D.)

In recent years this condition has become one of the most serious causes of economic loss to the poultry industry in America, in particular to the broiler industry where it is considered to be of greater economic importance than Newcastle disease or infectious bronchitis. Although mortality is variable and sometimes may be low, the serious effect on feed conversion and carcase quality results in large numbers of birds being rejected on marketing. Wilson (1954) states that the disease caused a loss of $20,000,000 in a few months in the broiler area of Delmarva. Since in many instances infection extends to the air sacs, the condition is also known as air sac infection. In fact one of the reasons for the rejection of poorly finished broilers is the appearance of thickened air sacs containing casts of caseous material.

The exact aetiology of the disease is still somewhat confused. Nelson, as early as 1936, described the agent as "A coccobacilliform body" but there is now little doubt that Nelson's organism is identical with the pleuro-pneumonia-like organism described by a number of American workers and in this country by Chu (1954). There also appears to be little doubt that the agent responsible for C.R.D. is similar to that present in turkey sinusitis (Markham & Wong, 1952; Van Roekel *et al.*, 1952; Grumbles *et al.*, 1953) Chu (1954), however, states that the P.P.L.O. may be isolated from fowls affected with a variety of disease conditions as well as from apparently healthy fowls, and he frequently found the organisms associated with infectious bronchitis, infectious laryngotracheitis and fowl pox. He suggests that although the exact pathological status of the organism is yet to be determined there may be a number of different types of P.P.L.O., some being sufficiently pathogenic to cause disease by themselves while others act only as secondary invaders. Certain workers also suggest the presence of a primary virus (Fahey & Crawley, 1954). The disease is commonest in young growing stock and the symptoms include coughing and sneezing clinically indistinguishable from Newcastle disease, infectious bronchitis, or even infectious coryza. Mortality, however, is usually higher than in the latter disease and may reach 50 per cent. Onset of egg producton is delayed and in adult birds there is a marked decline. The nasal exudate is similar to that seen in infectious coryza but accumulations in the sinuses are probably commoner and there is a greater tendency for chronicity and facial swelling. The incubation period may vary from 10 days to two to three weeks and the disease invariably runs a prolonged course, the birds remaining affected for as long as three to four months. Growth weight is markedly affected in growers and on autopsy lesions similar to those of infectious coryza are observed in the upper respiratory tract but in addition the curvical and abdominal air sacs are thickened and contain casts of yellow cheesy material, while thick yellow deposits are frequently found covering the heart, liver, and lungs. There is little doubt from recent work (Van Roekel *et al.*, 1954, and Cover & Waller, 1954) that the C.R.D. agent can be transmitted through the hatching egg from infected breeding flocks and Osteen (1954) refers to the outbreaks of the disease which had occurred following the use of Newcastle disease and infectious bronchitis vaccines presumably infected by the use of eggs from carrier flocks. There is little doubt that the disease tends to become endemic on certain farms presumably transmitted from adult carriers or even by contact between growers and subclinically affected carriers.

A number of antibiotics have been claimed to be effective particularly if given in the early stages of the disease, but most of the American workers are of the opinion that the only sound economic approach is to depopulate. However, we have found that streptomycin intramuscularly at a level of 25 mg. per kg. has been effective in certain field outbreaks, while in one outbreak the high-level feeding of oxytetracycline at a rate of 100 g. per ton had a spectacular effect on both symptoms and recovery in egg production; relapse, however, occurred some weeks later. Similar claims have been made for the high-level feeding of chlortetracycline (50 to 400 g. per ton). While the injection of oxytetracycline in oil (50 mg. per pullet) has been reported

by Carson *et al.* (1953 and 1954) to be equally effective. Van Roekel (1953), however, considers that the course and severity of experimental infection are altered little, if any, by antibiotic therapy. The control of C.R.D. is adequately reviewed by Crawley & Fahey (1955). The question of differential diagnosis and demonstration of P.P.L.O. in sinus exudate by staining methods and cultivation of the organism in tissue cultures is dealt with by Chu (*loc. cit.*) Adler (1954) refers to the use of a rapid slide agglutination test for the diagnosis of C.R.D. in the field which appears to specifically detect the presence of antibodies to P.P.L.O.

(3) Sinusitis in turkeys

Little need be said concerning this disease, since most workers now appear to be of the opinion that the causative agent is similar and possibly identical to P.P.L.O. associated with C.R.D. There is little doubt from the work of Van Roekel *et al.* (1952) that the agent responsible for C.R.D. can set up sinusitis in turkeys and that C.R.D. in chicken can be reproduced by material from infected turkeys. The same fact regarding carriers and egg transmission would appear to be applicable. The disease is relatively common in this country and of considerable economic importance, particularly in birds nearing marketing age. The symptoms which include coughing, sneezing, and head shaking are accompanied by an oculonasal discharge which accumulates in the sinuses causing marked swelling of the face, particularly below the eyes. Treatment, particularly in the early cases, can be effectively carried out by the intrasinal injection of streptomycin. Wilson, quoted by Blount (1955), has recommended the combination of 100 mg. streptomycin with 100,000 units penicillin in 2 c.c. of saline injected into the sinuses; while the dose may be boosted by the intramuscular injection of streptomycin for three to four days. Oxytetracycline is also claimed to be effective but in both C.R.D. and in sinusitis one may require to switch from one treatment to another. However successful the treatment may appear, carriers are probably established and depopulation should be attempted where practicable.

(4) Fowl cholera

Respiratory symptoms are occasionally seen in both acute and chronic fowl cholera. The odd bird in a flock, particularly in turkeys, occasionally shows difficult breathing while death may occur suddenly in a matter of 48 hours. Mortality in the chronic form is generally low and in cockerels may be accompanied by swelling of the wattles. On autopsy *Pasteurella aviscepticus* can usually be isolated from the solidified material in the wattles and from necrotic areas in the lungs. In turkeys treatment has been effectively carried out by the intramuscular injection of oxytetracycline in oil at a level of 25 mg. per kg.

(5) Pullorum disease

This condition should, of course, present no difficulties, for although the chicks may show difficult respiration, the autopsy findings of yellow-grey nodules varying in size from pin's head upwards, in the lungs, myocardium, liver, and gizzard are fairly diagnostic and there is little difficulty in the cultural isolation of *Salmonella pullorum*. Treatment can be effectively carried out by furazolidone at a level of 0.04 per cent. for 10 days (Williams Smith, 1955).

(6) Aspergillosis

Brooder pneumonia or pneumo-mycosis is relatively common in chicks, particularly where dusty and mouldy hay has been used for litter or chick box packing. The symptoms are similar to those described for *pullorum* disease and on *post-mortem* examination similar nodules are found in the lungs, but not in other organs, except occasionally in the air sacs. Diagnosis by cultural methods presents no problem. Although no medicinal treatment is known, Horton-Smith & Long (1952) showed penotrane to be a highly efficient fungicide and a proprietary form of this drug* has been used in some outbreaks to spray the litter, with apparently successful results. The use of this compound in aerosol form might be worthy of trial.

(7) Gapes

Infestation with *Syngamus trachea* may give rise to difficult respiration, mouth breathing, and coughing. The condition, however, is usually rare with the exception of outbreaks in pheasants and occasionally in turkey poults and requires little comment. Furthermore, diagnosis is simple. Treatment by the insufflation of barium antimonyl tartrate into an enclosed box containing the chicks is effective.

(8) Vitamin A deficiency or nutritional roup

This disease is by no means uncommon and in many instances results not from a gross deficiency in the diet but from over-dilution of the ration with home-grown cereal, or to faulty storage or deterioration of vitamin A when supplied as fish-liver oil. The condition responds rapidly to the addition of vitamin A. It mainly appears in growing chicks although symptoms may also occur in adult stock if the deficiency is sufficiently prolonged. In the early stages there is a thin, sticky discharge from the nostrils and eyes with the usual accumulation of caseous material in the sinuses, closely resembling infectious coryza and C.R.D. Caseous material, however, can usually be readily evacuated by pressure and the characteristic white colour of the exudate is almost diagnostic. Confirmation can usually be obtained by the presence of multiple raised white pustules in the pharynx and upper oesophagus.

(9) Fowl paralysis

Laboured breathing may be observed in cases of paralysis where the vagus or intercostal nerves are involved or where lymphoid infiltration has occurred in the lungs. Coughing, however, rarely occurs and there is no nasal exudate. *Post-mortem* examination readily demonstrates lesions in the nerves and the condition invariably occurs in individual cases.

* Fibrotan.

Cause	Name	Age	Incubation period	Duration	Spread	Mortality	Nasal discharge	Facial swelling	Trach lesio
I. Virus	1. Fowl pox	All ages, mainly adults	7–14 days	3–4 weeks	Slow	Variable but low 0–30%	In oculo-nasal form	Rare	Rare
	2. Infectious laryngo-tracheitis	Growers and adults	4–21 days	•2–3 weeks	•Rapid	•High, 30–90%	None	None	•Bloo clot
				†Months	†Slow	†Low, 5–10%			
	3. Newcastle disease	All ages	5–7 days	•Few days	•Rapid	•High, 100%	Slight	None	Non slig
				†Week or so in field	†Rapid	†High in chicks, 10–60%. Low or nil in adults			
	4. Infectious bronchitis	Chicks and adults	18–24 hours	5–21 days	Rapid	Chicks high, 60%. Adults low or nil	Slight in chicks only	None	Non slig
II. Bacterial	1. Infectious coryza (*Haemophilus gallinarum*)	Growers and adults	24–48 hours	10–14 days	Rapid	Slight, 0–10%	Profuse	Slight to severe	Non
	2. Chronic respiratory disease (P.P.L.O.)	Growers mainly	10–20 days	Months	Slow	Variable to high, 10–50%	Profuse	Slight to severe	Non
	3. Infectious sinusitis (P.P.L.O.)	Turkeys all ages	2–4 days	Weeks to months	Rapid	Variable	Profuse	Severe	Non
	4. Pullorum disease (*S. pullorum*)	Chick	3–5 days	Few days	Rapid	Usually high. 20–60%	No	No	No
	5. Fowl cholera (*P. avisepticus*)	Adults and turkeys	2–3 days	•Few days	•Rapid	•High, 80%	Unusual	•No	No
				†Months	†Slow	†Low, 5%		†Swollen wattles	
III. Fungal	Aspergillosis	Chick	2–4 days	Months	Rapid	Variable to high, 10–50%	No	No	No
IV. Nutritional	Avitaminosis "A" (nutritional group)	Growers	—	—	—	Usually low, 5–10%	Yes	Yes	No

• Acute. † C

Summary

I apologise for the superficial way in which I have dealt with the differential field diagnosis of respiratory diseases, but I am sure you will appreciate that the multiplicity of conditions all giving rise to almost similar symptoms presents a nearly insuperable problem for the routine laboratory worker or field adviser. Whereas certain diseases such as gapes, paralysis, aspergillosis, pullorum disease, cholera, fowl pox, and in certain instances infectious laryngo-tracheitis can be fairly readily differentiated, others of far greater importance such as Newcastle disease, infectious bronchitis, infectious coryza, and C.R.D. remain a very confused issue, unless facilities are available for the diagnostic techniques referred to. Unfortunately, as I have indicated, most of these techniques are too laborious for the routine worker and frequently too time-consuming to be of practical value to the stock owner. Probably in most instances the best procedure is to regard all outbreaks accompanied by respiratory symptoms as potential Newcastle disease and having eliminated that condition to proceed step by step either by laboratory methods or by field trials. At least by *post-mortem* examination alone, one can eliminate paralysis and *Syngamus* and by cultural methods *pullorum* disease, cholera, and aspergillosis. The diagnosis of both pox and tracheitis by direct transmission is relatively simple, but having eliminated those and Newcastle disease one is still left with the often nearly insoluble problem of the accurate differentiation of infectious coryza, C.R.D., and infectious bronchitis. Therapy trials on small groups can occasionally give a helpful lead since only coryza responds to sulphonamides, while C.R.D. and bronchitis may possibly be differentiated on the clinical evidence. The position, however, is far from satisfactory.

Tracheal lesions	Breathing	Other symptoms	Carriers	Diagnosis	*Post-mortem* examination	Control
Rare	Normal exc. in diphtheritic type	Pox lesions wattles, comb, eyelids. Membranes mouth, tongue, palate	No	Clinically inoculated chick embryos or susceptible fowl	As for symptoms. Membranes adherent	Pigeon pox or fowl pox live virus vaccines by scarif. or stab.
*Blood and clots	Coughing, rattling, outstretched head and neck, open beak. Coughing blood splashes in pen. Cyanosis and suffocation		Yes, over one year	As above	*Blood and casts in trachea †Necrotic material larynx, trachea	Live virus vaccine applied to cloacal membrane in some countries
None or slight	Coughing, sneezing	*Coughing, cyanosis. Green diarrhoea. Sudden death †Drop in egg yield. Abnormal eggs. Nervous symptoms (head and neck)	Probably not	H.I. test. Isolation virus in embryos and neutralisation tests. Inoculated susceptible fowl	Haemorrhage Proventriculus ulcers	Scheduled disease in G.B. var. vaccines in other countries
None or slight	Sneezing. Difficult breathing. Crackling in chicks	Drop to cessation of egg yield. Grossly abnormal shelled eggs in adults	Yes	Embryo inoculation dwarfing. Serum neutralisation	Slight necrotic material or clouding in air sacs in chicks	Live vaccines. Spray application
None	Sneezing and head-shaking, closed eyes	Poor condition. Loss in egg and weight gain	Yes	Clinically smears and cultures	Caseous material in sinus and mouth yellow and loose	Sulphonamides. ? Furazolidone
None	Coughing and rattling	Loss in weight gain and egg yield	Yes	Clinically, smears, tissue, cultures	Caseous material in sinus air sacs, heart and liver	Antibiotics i.m. and dietary (high level)
None	Sneezing and difficult breathing	Swollen face. Emaciation	Yes	As above	Caseous material in sinus and occasional air sacs	Antibiotics i.m. intrasinus and dietary (high level)
No	Difficult breathing	Diarrhoea	Yes	Cultures	Nodules ht. lungs, liver, etc.	Blood test furazolidone
No	Laboured	Swollen wattles, cyanosis, sudden death	Yes	Cultures	Necrosis, lungs, caseous material in wattles	Oxytetracycline i.m.
No	Laboured	None	No	Cultures	Nodules in lungs and air sacs	? Penotrane on litter
No	Laboured	Poor feathering. Poor growth. Emaciation	—	Clinically	White cast in eyes and sinus pustules, gullet and oesophagus	Vitamin A

† Chronic.

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Some Observations on Respiratory Disease in Poultry

BY

F. T. W. JORDAN

Department of Veterinary Preventive Medicine, University of Liverpool

IN the opinion of poultry keepers and farmers in Lancashire, Cheshire, and Shropshire, the incidence of colds or coryza in poultry has increased during the last few years. During the last 18 months I visited a number of farms where outbreaks were occurring and found the clinical picture followed the same pattern on all of them. The clinical symptoms, pathology, epidemiology, and some of the possible aetiological agents were studied over a 12-month period on one of these farms. This farm carries about 9,000 adult stock and rears about 13,000 chicks to eight weeks of age and about 8,000 birds to six months of age. New stock is introduced only in the form of hatching eggs which are hatched on the farm. A variety of pure breeds are kept; Rhode Island Reds, Brown Leghorns, Light Sussex, Indian Game, and also cross breeds. Orthodox methods of rearing are practised (paraffin, electric, and infra-red brooders). Some of the chicks are put in hay-box brooders from four to eight weeks of age while others are provided with heat until six to eight weeks old. At this stage male and female birds are segregated and then reared on free range or in fold units. A proportion of the growers are sold but those that are retained are made up into breeding flocks at about six months of age for the production of hatching eggs. Most of these are housed semi-intensively but some are kept in straw-yards and folds, and a smaller number on free range and on deep litter.

Facilities for clinical observation were good, and over the past 18 months the carcases of all poultry dying or killed because of ill-health have come for autopsy. There was very little culling, and all birds culled because of ill-health came for autopsy.

Two clinical forms of respiratory disease were seen. Firstly, a condition of coryza, usually epidemic in nature, mainly affecting growers, rarely fatal, and in which *post-mortem* lesions consisted chiefly of a mild catarrhal inflammation of the upper respiratory tract, nares, trachea, and sinuses. Secondly, a sporadic condition mainly affecting mature birds, which was always fatal and was shown either by respiratory distress, or by merely a progressive depression and emaciation. At autopsy, lesions were found in the trachea, lungs, or air sacs.

The only other instances of respiratory disease were restricted to a few cases of tumours, sporadic aspergillosis in chicks, and obstruction of the trachea with foreign bodies.

Respiratory Disease—Type 1

This condition was known to exist on this farm for many years, and the clinical signs were those typical of coryza. There was always a nasal discharge, which was at first thin, watery and colourless, and later became tenacious and muco-purulent and sometimes had a foul odour. Other symptoms included head shaking, coughing, sneezing, and occasional rattling or gurgling respiratory noises. Distension of the infra-orbital sinus was seen infrequently. Depression and anorexia were uncommon and occurred only in the early stages of the disease. Most birds remained bright throughout the course of the disease. Morbidity was high but mortality was negligible.

The age of affected birds varied, but the condition was not seen in birds under eight or nine weeks old. Birds were usually first affected at three to five months, and in birds of this age the condition was widespread throughout the farm.

Less frequently, the epidemic was seen in older birds, for example, in birds of six to seven months within four to five weeks of transfer to breeding pens, and there were also two outbreaks in birds of 10 to 11 months. Besides this epidemic type of coryza, sporadic cases were seen in flocks of mature birds, but it is not known whether they were new cases or a recurrence of previous infection.

The disease occurred in both sexes, in all breeds and cross breeds, and with all types of housing and management. Feeding and cleanliness did not appear to be significant factors and the standard of both on this farm was high.

Although initial outbreaks were encountered in almost every month of the year, the disease was most common in June, July, and August, but this may be associated with the fact that there were more growers on the premises at that time.

During the course of a coryza epidemic in a particular flock the number of birds showing symptoms varied from day to day, and in some flocks entirely disappeared for a while, only to reappear later and then disappear again.

Detailed observations were made on the intermittent presence or absence of coryza in 12 small flocks, each of about 25 birds, kept in fold units. The age of the flocks varied from three to five months and they were all observed over the same 10-week period (August to November), the observations commencing within a few days of the initial signs of the outbreak. Every bird was observed for clinical signs of coryza at least twice weekly, and every day for the first month.

The intermittent nature of the disease in these flocks can be seen from Fig. 1, where the black lines indicate the presence of clinical infection and the spaces indicate its absence. There appeared to be no uniform pattern of disease running through the flocks. One of the 12 flocks, consisting of 27 birds four months old, was observed over a longer period (25 weeks), during which time each bird was handled and examined for the presence of coryza, including applying gentle pressure to the nostrils. For the first month the birds were examined every day, and thereafter at least twice weekly. By this means the spread of disease in a flock, the variation in the number of birds showing clinical signs from time to time, and the presence or absence of the clinical disease in the individual birds could be seen. The spread of infection is shown in Fig. 2, where it will be seen that it was at first quite rapid, 22 of the 27 birds being affected within 16 days of the first evidence

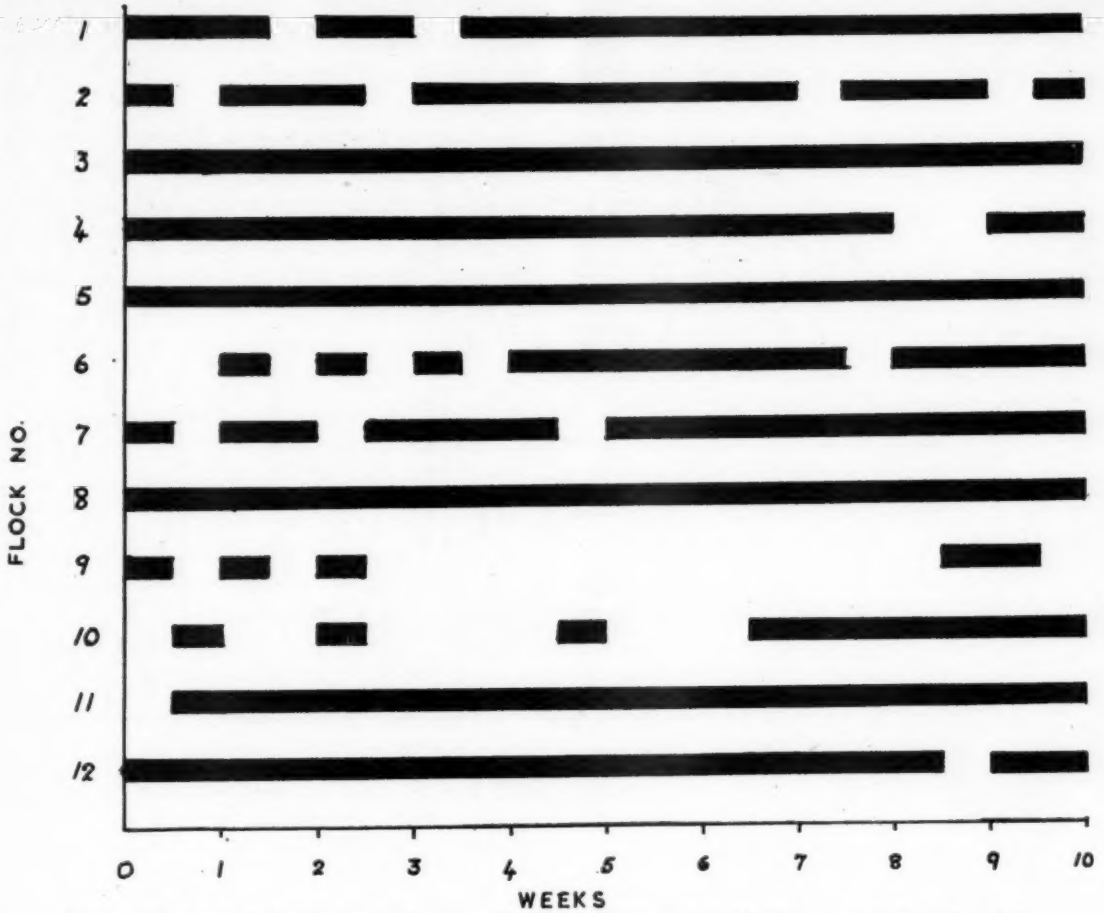


FIG. 1.—Incidence of clinical signs of coryza (shaded black) as seen in 12 flocks over a 10-week period.

of disease. Eventually the whole flock was affected, the last bird showing clinical signs 14 weeks after the disease was first noticed. The fluctuation in the number of birds showing clinical signs of coryza is also shown. Sixteen weeks from the commencement of the outbreak all members of the flock were clinically normal. This apparent recovery lasted for nearly a fortnight. During the succeeding 10 weeks clinical cases recurred, their presence alternating with periods of freedom from the clinical disease. The high peaks at approximately 14, 19, and 21 weeks coincided with the only three periods of dense fog.

Clinical evidence of infection in individual birds, as in the flock, followed an intermittent pattern which varied from bird to bird, and Fig. 3 illustrates the course of disease in two birds. Bird A showed symptoms continuously for a long period followed by six short periods, while B showed signs of disease on only two occasions and in each case they persisted for only a few days.

Respiratory Disease—Type 2

The condition occurred sporadically. Some birds showed a gasping type of respiration even at rest, gurgling respiratory sounds, coughing and depression, and occasionally foul breath. Nasal discharge or swelling of the infraorbital sinuses was rare. Other birds showed merely, progressive depression and emaciation. *Post-mortem* examination of birds which showed respiratory symptoms revealed obstruction of the larynx, trachea, or bronchi, by caseous or semi-solid pale yellow exudate (Fig. 4), sometimes together with tenacious mucus. Birds which had shown no respiratory distress during life showed a similar exudate, but it caused no macroscopic obstruction and was confined to the lungs or air sacs (Fig. 4C). Thickening of the air sac walls was often seen. Most carcases were emaciated but in some, where caseous exudate was found obstructing the trachea or primary bronchi, death had presumably occurred from asphyxia before there was time for loss of condition (Fig. 4A).

Such respiratory lesions were assumed to have been the cause of death when no evidence of any other fatal condition was revealed at autopsy.

A few birds dying from other causes, such as egg peritonitis and visceral gout, also showed lesions in the respiratory tract at autopsy, but these were not

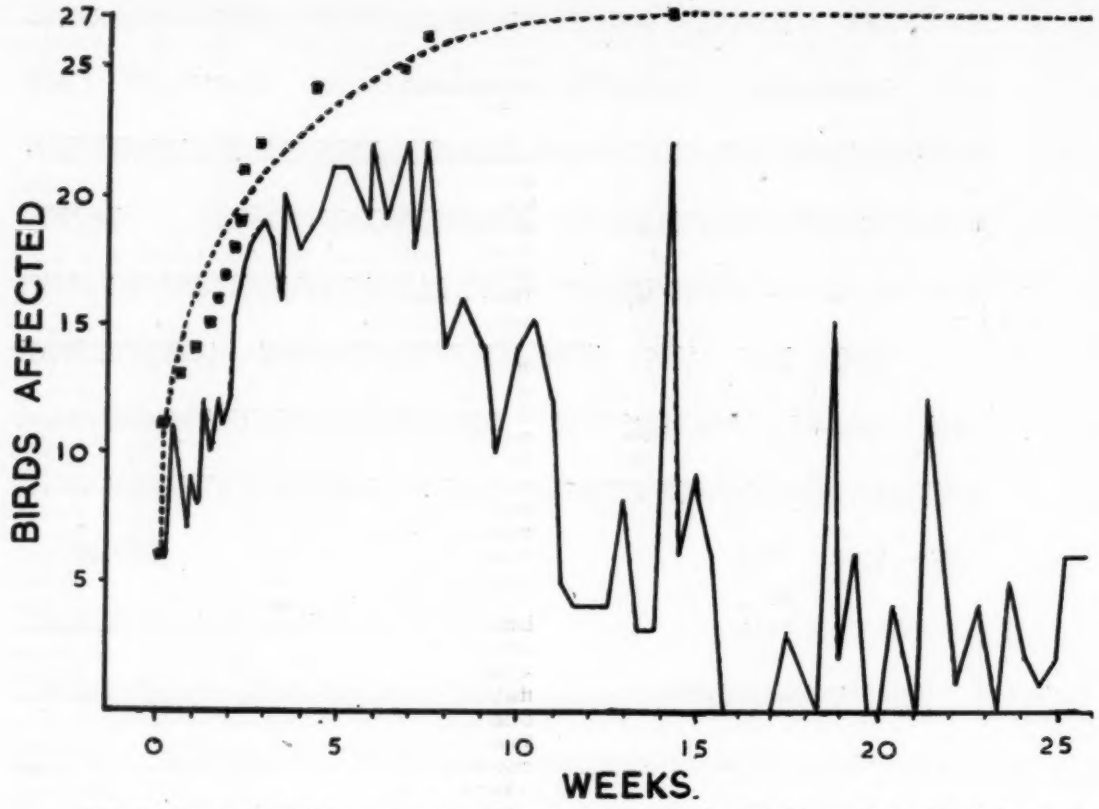


FIG. 2.—Spread of clinical coryza and variation in numbers of birds affected in one flock of 27 birds.
Rate of spread of infection - - - - - - - - - - No. clinically infected ————————

included among deaths attributed to respiratory disease. This means that birds affected with lesions of respiratory disease were more numerous than the number of deaths attributed to this disease.

The incidence and the significance of the fatal form of respiratory disease can be seen from the *post-mortem* records. During the 12-month period November 1st, 1954 to October 31st, 1955, a total of 1,364 poultry carcases were examined from this farm and 154 (11.2 per cent.) of these birds were considered to have died from this fatal type of respiratory affection (Table I). Deaths from all causes and deaths from this type of respiratory disease were classified in relation to age and sex.

Only 2.9 per cent. of deaths in birds under six months were due to respiratory disease, whereas for birds older than this the incidence ranged from 13.9 per cent. to 21.4 per cent. In all age groups respiratory disease caused a higher proportion of deaths in relation to population at risk in males than in females. The distribution of the lesions is shown in Table II where they are classified according to the

TABLE I

TOTAL DEATHS AND DEATHS CAUSED BY RESPIRATORY DISEASE, TYPE 2

Age (months)	Number of deaths						Respiratory deaths per cent. of total
	From all causes			Attributed to respiratory disease			
	♂	Total	♀	♂	Total	♀	
0– 6	183	584	401	10	17	7	2·9
6–12	43	348	305	26	57	31	16·4
12–18	25	205	180	18	44	26	21·4
18–24	5	79	74	1	11	10	13·9
24 and over	0	148	148	0	25	25	16·9
All ages ...	256	1,364	1,108	55	154	99	11·2

TABLE II

DISTRIBUTION OF LESIONS IN THE RESPIRATORY SYSTEM—154 DEATHS FROM RESPIRATORY DISEASE, TYPE 2

Site	Number of carcases in which lesions found	
	Restricted to one site	Found at site but not restricted to it
Larynx, trachea, bronchi...	24	82
Lungs	10	88
Anterior air sacs—cervical, clavicular and thoracic ...	11	84
Abdominal air sacs ...	5	54

part of the respiratory tract affected. There was no particular sex or age difference. It can be seen that 24 birds (15 per cent.) died with lesions confined to the larynx, trachea, or primary bronchi, and that the abdominal air sacs were less-frequently affected than either of the other three sites shown in the table.

Aetiology

Newcastle disease was excluded by H.I. tests and Vitamin A deficiency by the estimation of Vitamin A levels in the liver and in some cases in the blood of affected birds. Fowl pox and infectious laryngo-tracheitis were not seen clinically in their typical form but they were not specifically eliminated by examining for the presence of the virus. Infectious bronchitis virus was also not looked for.

A total of 65 samples of exudate from various sites in the respiratory tract of 40 birds was examined culturally for each of the following organisms:—pasteurella, haemophilus, fungi, and pleuropneumonia-like organisms (P.P.L.O.). Some of the birds were alive and affected with coryza (respiratory disease—type 1) while others were seen at autopsy to be affected with the second type of respiratory disease. In the latter case a little exudate was removed asceptically from the affected site and sown on to blood agar, chocolate agar, malt extract agar with penicillin and streptomycin, and on solid and semi-solid Edward's medium.

In the case of living birds, swabs from the nasal and tracheal mucus, and exudate aspirated aseptically from the infraorbital sinuses, were sown on to the respective media. Except for solid Edward's media, which was incubated for a week, media were incubated at 37° C. for four days and then discarded. Subcultures from semi-solid Edward's media were made on to solid Edward's medium at 48 and 72 hours, and these were incubated and examined for a further week if necessary.

Table III shows the organisms isolated from various sites. Neither pasteurella nor haemophilus was isolated. *Aspergillus fumigatus* was isolated four times each from the caseous material in the trachea, lungs, and air sacs. P.P.L. organisms were isolated on 17 occasions from mucus in the trachea, three times from mucus in the infraorbital sinuses of living birds showing coryza, and twice from nasal exudate. This organism was also isolated from caseous exudate from two birds that died of respiratory disease type 2.

The value of these observations is limited because the possible presence of such diseases as the mild form of infectious laryngo-tracheitis and infectious bronchitis was not eliminated. The aetiological agents involved in the two types of respiratory disease on this farm must therefore remain a matter for conjecture, but it seems unlikely that one and the same causal organism could produce two such different epidemiological, clinical, and pathological conditions as coryza in young birds and the lesions seen in the trachea, lungs, and air sacs of older birds. Although P.P.L. organisms have been isolated frequently from birds affected with coryza and from two birds affected with the second type of respiratory disease, their exact significance is not clear, for Chu (1954) has shown that they can be isolated from outbreaks of infectious bronchitis, infectious laryngo-tracheitis, and also from apparently healthy fowls. *Aspergillus fumigatus* may well be a secondary invader because the organism is known to be present in a proportion of normal lungs (Jordan, 1954). Whatever the aetiology may be the

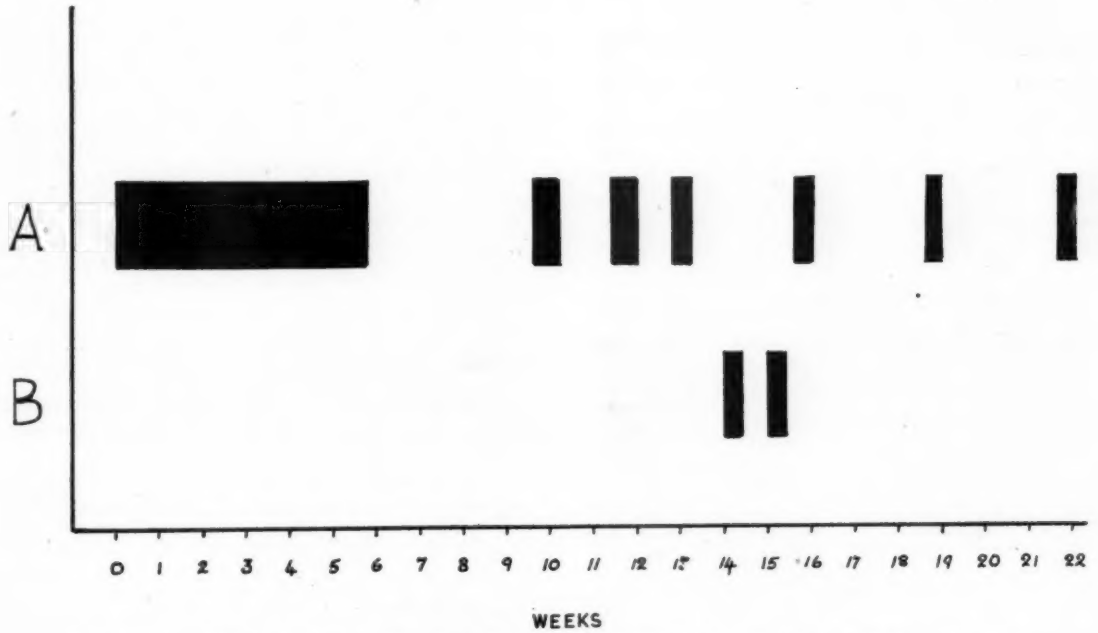


FIG. 3.—Intermittent nature of clinical signs of coryza (shaded black) as seen in two birds from one flock.

TABLE III
ISOLATION OF ORGANISMS FROM CASES OF RESPIRATORY DISEASE

Site and material cultured	Number of samples examined	Organisms isolated				Negative
		Pasteurella	Haemophilus	*A. fumigatus*	P.P.L.O.	
Nasal exudate—mucus* ...	9	0	0	0	2	7
Mucus in infraorbital sinus* ...	4	0	0	0	3	1
Tracheal mucus*	25	0	0	0	17	8
Caseous exudate—trachea† ...	9	0	0	4	1	4
„ „ —lungs† ...	5	0	0	4	0	1
„ „ —air sacs† ...	13	0	0	4	1	8

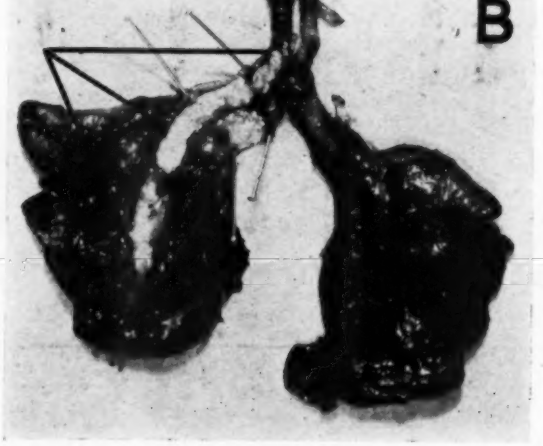
* Respiratory disease, type 1 ... 29 birds. † Respiratory disease, type 2 ... 11 birds.

second type of respiratory disease was of considerable importance since it caused more than 10 per cent. of total deaths, nearly one fifth of the deaths in birds between 6 and 18 months.

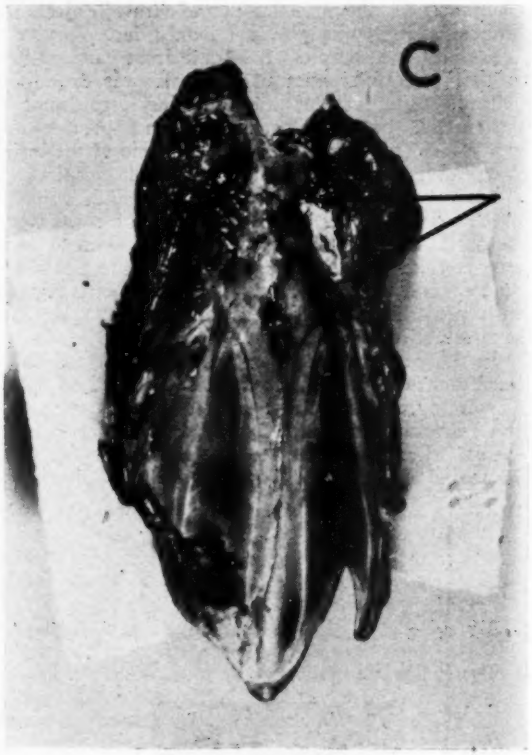
The first type of respiratory disease was characterised by high morbidity and negligible mortality, but since the disease was mild it was impossible to determine its economic significance in the absence of control-uninfected birds.

FIG. 4.—Respiratory Disease—Type 2.

A. Caseous exudate obstructing upper trachea—good condition as shown by sternum.



B. Caseous exudate extending from syrinx into lung.



C. Caseous exudate in right clavicular air sac (visceral surface of sternum).

Acknowledgments.—I wish to thank Miss J. Berry, Mr. L. G. Kelley, and Miss B. H. Messum for technical help.

I am grateful to Dr. J. Lowe of the Biochemistry Department of this University for examination of blood and liver for Vitamin A content, and to Miss M. A. Gentles for the photographs.

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In Brief

THE TREATMENT OF POISONING WITH LEAD AND OTHER HEAVY METALS

CONVENTIONAL treatment of acute poisoning with heavy metals has been confined largely to the relief of symptoms such as colic, frenzy, etc., together with measures to precipitate unabsorbed metal in the alimentary canal, and to facilitate the deposition in the skeleton of absorbed metal.

Methods of combating the toxic action of heavy metals *after absorption*, as for instance with dimercaprol (B.A.L.) or citrates, have been found unsatisfactory. Recently, however, a new group of chemical agents has been investigated with success, especially in lead poisoning. These are chelating agents, in particular derivatives of ethylene-diamine-tetra-acetic acid (E.D.T.A.). Chelating agents have the property of binding divalent metal ions to form stable, soluble complexes which are non-ionised and thus virtually free of the toxicity of the metal concerned.

E.D.T.A. itself is toxic to animals since it removes calcium and other ions from body fluids, but if given as the calcium-E.D.T.A. complex there is no risk of hypocalcaemia. The affinity of E.D.T.A. for various metals varies considerably, being high for heavy metals such as lead, intermediate for calcium, and lower for magnesium. Metals " compete " for the chelate in proportion to this affinity; thus lead displaces calcium from E.D.T.A. and calcium displaces magnesium.

On administration of calcium E.D.T.A. to an animal whose blood and tissues contain ionic lead, the calcium is displaced from the chelating agent and non-toxic lead chelate formed instead, with consequent relief of symptoms and rapid excretion of the lead chelate. This removal of lead is much more efficient when present in the soft tissues than when incorporated in bone, and the efficacy of this treatment of lead poisoning is inversely proportional to the delay in commencing treatment.

Ca-E.D.T.A. may be given by almost any route and its use has been studied in man, laboratory animals, dogs, cats, horses, and calves. Its most obvious application in veterinary practice is in the treatment of acute lead poisoning in which it should be given repeatedly for several days, but it may also be useful in intoxications with other metals such as mercury, iron, copper, chronium, beryllium, and radio-active elements.

R. J. FITZPATRICK (November, 1955).

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News and Comment

CONGRESS HOTEL ACCOMMODATION

There is a small amount of accommodation still available in hotels where " block " bookings have been made on behalf of the Association. The time is running out when these rooms can remain reserved by the hotels concerned, and members who have not yet secured accommodation are strongly advised to do so immediately. Application for details of the hotels where rooms are still available should be made to Mr. J. H. McGhee, 46, Coten End, Warwick, who is hon. secretary to the Provisional Committee.

ANIMAL HEALTH TRUST AWARDS

The Animal Health Trust announces the following list of successful 1956/7 applicants for Research Training Scholarships:—

Miss J. A. Bailey, B.SC. (ZOOLOGY), who is to work upon problems of parasitology at the Imperial College, London.

Mr. R. S. Anderson, B.V.M.S., M.R.C.V.S., who is to work at the School of Veterinary Medicine in the University of Glasgow studying the problem of metabolic diseases in animals, and in particular the electrolyte/water balance in animal tissues.

Mr. J. W. Boyd, M.R.C.V.S., who will study Part II of the Tripos Examination in Pathology at Cambridge preparatory to undertaking research into metabolic diseases of animals.

Mr. Muhammad Irfan, F.S.C., B.V.SC., M.R.C.V.S., who is to study the blood picture of dogs, with particular reference to tumours and leucosis conditions, at the Royal Veterinary College, London.

Miss S. R. Wooldridge, B.A. (OXON), daughter of Dr. W. R. Wooldridge, founder of the Trust. Miss Wooldridge is to study problems connected with the effects of radiation upon cells in tissue culture, paying particular attention to changes concerned with cellular division and multiplication and their late application to cellular pathology. The work is to be carried out in the Radiotherapy Laboratories at Cambridge where valuable research is being done on cancer in man and animals.

FOOT-AND-MOUTH DISEASE

The area described in the schedule below is declared to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease:—

COUNTY OF GLOUCESTER

The Petty Sessional Divisions of Coleford and Newnham.

Petty Sessional Division of Newent. The parishes of Huntley, Tayton, Tibberton, Rudford, Newent, Oxenhall, and Kempley, and so much of the parish of Dymock as lies to the south and west of the road running from Much Marcle via Dymock to Newent including the said road.

Petty Sessional Division of Gloucester. The parish of Churcham.

Petty Sessional Division of Lydney. The parishes of Alvington, Aylburton, and Lydney.

COUNTY OF HEREFORD (Petty Sessional Division of Ross). The parishes of Welsh Bicknor, Goodrich, Walford, Hope Mansel, Bridstow, Ross Urban, Ross Rural, Weston-under-Penyard, Lea, Aston Ingham, Linton, Brampton Abbotts, Upton Bishop, Foy, Brockhampton, How Caple, Yatton, Sollers Hope, Ganarew, Llanrothal, Welsh Newton, Whitchurch, Llangarren, Marstow, Tretire-with-Michaelchurch, Hentland, Peterstow, Sellack, King's Caple, Ballingham, Harewood and Pencoyd, and so much of the parish of St. Weonards as lies to the east of the road running from Much Birch via St. Weonards to Welsh Newton including the said road.

Petty Sessional Division of Ledbury. So much of the parish of Much Marcle as lies to the south of the road running from Sollers Hope via Much Marcle to Dymock including the said road.

COUNTY OF MONMOUTH. The Borough of Monmouth and the Petty Sessional Division of Monmouth.

Petty Sessional Division of Trelleck. So much of the parish of Trelech United as lies to the east of the main road running from Monmouth to Devauden including the said road.

LICENSING OF DUAL-PURPOSE VEHICLES

The Parliamentary Committee of the Royal College, having received a number of enquiries as to whether vehicles of the shooting-brake type require to be licensed as " goods " vehicles, sought the view of the appropriate Ministry. The reply received included the following:—

" We are advised that the use of a utility vehicle by a professional man to carry articles used in connection with his profession is use ' for or in connection with a trade or business.' If, however, such articles are small and are normally carried in an attaché or brief case we consider that they could reasonably be disregarded for taxation purposes so that the vehicle could then be classified in the same way as a private car. Where other bulkier goods are carried in connection with the owner's profession the vehicle would, of course, be taxed at the ' goods ' rate. All registration and licensing authorities are fully aware of these provisions and we see no reason for differentiation in treatment as between one authority and another."

The solicitors to the Royal College were asked whether any useful purpose would be served by conducting a test case on the interpretation of the current licensing regulations regarding the distinction between " goods " and private vehicles. In the course of their reply the solicitors wrote:—

" We observe that in practice the higher rate is not demanded where a professional man can carry articles used in connection with his profession in an attaché or brief case, but the higher rate of tax is demanded where bulkier goods are carried.

" In our view no useful purpose would be served in seeking to challenge this arrangement by means of a test case since, on the strict interpretation of the law, the higher rate of duty should be paid however small or easily portable are the articles which the veterinary surgeon carries for the purpose of his profession and, therefore, the only effect of challenging the arrangement might be to endanger the very sensible concession which the Treasury make."

Fuller details of this correspondence will be published in the forthcoming Supplement.

ERRATUM

We regret that in our issue of July 21st, the qualifications of Mr. R. J. Thomas, co-author of the paper " Some Observations on Nematodirus Disease in Northumberland and Durham " were wrongly quoted. They are B.Sc., M.Sc., M.R.C.V.S.

MR. MURDO FERGUSON

Mr. Donald Campbell writes:—

His many friends, whether in the ranks of the profession or among the general public, will have learned with grief of the sudden death of Murdo Ferguson at the early age of 38 years. His kindly, genial disposition attracted people from every walk of life. He met them all with the same sense of sympathy and understanding, and there can be very few men who have enjoyed such universal esteem as he. His untiring efforts towards perfection in the diagnosis and treatment of animal disease were a constant feature of his day's work. His ability as a practitioner, and his desire to attain the highest possible ethical standard, gave him an honourable status in the profession, and among the agricultural community of the West Riding of Yorkshire.

Those of us who had the privilege of close association with Murdo, either during college days, or in post-graduate life, sadly mourn his loss, the loss of a real friend. As a partner, one could not wish for anyone more reliable, more understanding, or more sincere.

The memories of many pleasant times spent together will always remain with me. Murdo's door was open to all, and what a happy home it was until this cruel blow came. Both he and Irene, his wife, were never happier than when looking for extra chairs to accommodate the many friends who called on them.

To Irene, in her great sorrow, and to their dear little son, Ian, we extend our heartfelt sympathy.

Mr. H. M. MacMillan writes:—

His many friends must have been shocked to hear of the sudden death of Murdo Ferguson. I had the privilege of knowing him well before the war when we were students together at the Glasgow Veterinary College. His ready wit, smiling face, and charming personality endeared him to all with whom he came in contact.

He was a keen and fluent Gaelic scholar and liked nothing better than an evening at the " Highlanders' Institute " or a " Ceilidh " at a friend's house. What happy memories this recalls.

An able and conscientious practitioner, he rapidly earned himself a high reputation in the profession and in the farming community of the West Riding of Yorkshire.

To his wife Irene and family we extend our deepest sympathy. May they be comforted in the knowledge that in the " Tir nan Og " sorrow and grief are unknown.

He was a Highland gentleman—better rank no man could wish to be.

Mr. J. David Copley writes:—

I first met Murdo Ferguson shortly after he arrived in Keighley as I was at that time an undergraduate gaining experience in that practice. As a result of our meeting I gained an excellent and sympathetic guide to many branches of professional work and furthermore a staunch friend. The concise and accurate way in which he explained cases to me at that time not only showed his willingness to help younger people, but also how deep was his own interest in this work which had become his life and to which he gave his energies unsparingly.

After qualifying and settling in a neighbouring practice, my friendship with him grew with regular—but all too infrequent—visits. On these occasions the conversation invariably turned upon professional topics. Murdo was always seeking to achieve a proper perspective in his professional life, and thus to give the utmost possible service to others.

Although he had been in Keighley so short a time, first as an assistant and then succeeding to partnership, the respect and admiration which he commanded in all quarters of the community, due to his ever cheerful disposition and to his unflagging zeal for his work, was very great indeed. Now, as he was barely settled in his work, before his mark was made but not before his presence was appreciated, this man of such undoubted promise is taken from us just as his life was to bear fruit. We, his friends and professional colleagues, extend our deepest sympathy to Mrs. Ferguson and her son Ian in this tragic loss.

MR. REGINALD W. HALL

Mr. D. G. Morgan writes:—

With the death of Mr. R. W. Hall the South Wales Division loses one of its founder members, a past president, and one of its most faithful council members.

The funeral took place on Tuesday, July 31st, a service being held at All Saints' Church, Barry, followed by cremation at Thornhill Crematorium, Cardiff. There was a large gathering at the Church representative of local government, the agricultural community, and the Barry Athletic Club. The profession was represented by Mr. A. G. Beynon, Col. J. B. Griffiths, Messrs. D. G. Llewelyn, D. R. Allen, A. J. Woods, R. H. Packer (Mr. Hall's partner), D. O. Packer, W. E. O. Davies, E. T. Watts, and D. G. Morgan (representing the South Wales Division).

Colonel J. B. Griffiths writes:—

Mr. Reginald William Hall suffered a sudden heart attack on July 28th, 1956. He was in his 82nd year. Hall came from yeoman North Devon stock which endowed him with a sound appreciation of animals, and indomitable courage. He was educated at Cranleigh School and qualified at London in 1897. On graduating he returned as assistant to the Penhale practice at Barnstaple, where he remained until the following year, and then set up in practice on his own account at Barry, Glamorgan.

Hall soon established himself in the confidence of the farmers in the adjacent Vale of Glamorgan, and for the next 40 years ran one of the largest single-handed practices in the country. Early in the century he became one of the first veterinarians in the country to receive a part-time appointment under the Barry local authority as meat and dairies inspector, and this appointment he retained until some eight years ago. He was also a local veterinary inspector under the Glamorgan County Council and Ministry of Agriculture and Fisheries for many years.

Hall was joined in practice by his partner Mr. R. H. Packer in 1940, and only retired from active work at the end of last year, when he reached the age of 81. He was a keen sportsman all his life and kept an active interest on the bowling green up to the day of his death.

PERSONAL

Death

GLYN.—On August 7th, 1956, David, aged 10 years, dearly loved elder son of Christine (*née* Wortley) and Michael Glyn, M.R.C.V.S.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

FERGUSON, Murdo, 16, Devonshire Street, Keighley, Yorkshire. Graduated Glasgow, December 12th, 1950. Died suddenly July 24th, 1956, aged 38 years.

COMING EVENTS

September

12th (Wed.). Meeting of the North Wales Division, B.V.A., at Leahurst Veterinary Field Station, 11.30 a.m.

16th to 22nd (Sun. to Sat.). 74th B.V.A. Annual Congress at Royal Leamington Spa.

November

22nd and 23rd (Thurs. and Fri.). B.V.A. Conference on Supplements and Additives in Animal Feedingstuffs at the Victoria Halls, Bloomsbury Square, London, W.C.1.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ches. Lawton Hall Farm, Church Lawton, Stoke-on-Trent (Aug. 7).

Derby. Grange Farm, Newton Solney, Burton-on-Trent (Aug. 8).

Leics. Bondman Hayes Farm, Ratby (Aug. 8).

Lincs. The Mill Farm, Claypole (Aug. 10).

Foot-and-Mouth Disease

Glos. Meend Farm, Woodside, Ruardean (Aug. 8); Ruardean Woodside, Ruardean; Dunkleys Corner, East Dean; Jovial Colliers, Ruardean Woodside; The Branch, Drybrook; Glenarda, Ruardean Woodside; Buffins Orchard, Morse Road, Drybrook and Ruardean Hill, Drybrook; Wayside, Harrow Hill and The Nag's Head, Ruardean Hill, Drybrook; Bilson Green, Cinderford; Plump Hill, Mitcheldean (Aug. 10); Forestry Enclosure, Harry Hill, Drybrook; Field at Pludds, Ruardean; Field opposite Memorial Hall, Ruardean; Meadow at rear of Rock House, Ruardean Hill; Smithers Cross, Ruardean; The Nest, Longhope; Sunnap Cottage, Harrow Hill, Drybrook (Aug. 11); Sunnyside, Harrow Hill, Drybrook; Woodside, Ruardean; Harrow Hill, Drybrook; Gaggs Flat, High Beech, Ruardean; Harrow Hill and Devon House, Morse Road, Drybrook; Lodge Meadow, Chestnuts, Popes Hill, Newnham; The Poplars, Popes Hill, Newnham (Aug. 12).

Hereford. Large Barn Farm, Walford (Aug. 10).

Fowl Pest

Essex. Ardmore, London Road, Billericay (Aug. 8).

Lancs. Farmers' Arms Allotments, Heskin, Chorley; Moss House Farm, Preston Road, Charnock Richard, Chorley (Aug. 7); Silvester Farm, Washington Lane, Euxton, Chorley; Bridge Farm, Coppull, Chorley (Aug. 9); Robin Hood Farm, Ridley Lane, Croston, Preston (Aug. 10).

Lincs. Newhouse Farm, Dogdyke; Hawthorne Hill, Dogdyke (Aug. 7); 103c, Wyberton West Road, Boston; George Avenue Allotments, Skegness (Aug. 8); 103b, Wyberton West Road, Boston (Aug. 10).

Sussex. Hendall's Farm, Maynard's Green, Horam (Aug. 9).

Swine Fever

Dorset. Hillcrest Farm, Fontmell Magna, Shaftesbury (Aug. 7); Cadmans, Marnhull, Sturminster Newton (Aug. 9); Keepers Cottage, Clyffe Woods, Tincleton, Dorchester (Aug. 10).

Cornwall. Trennick Farm, Truro (Aug. 13).

Lancs. Oaklands, Ashley Lane, Goosnargh, Preston (Aug. 10); Sunnyfields Farm, Moor Lane, Ince Blundell, Hightown, Liverpool (Aug. 13).

Lincs. Piggeries at St. Hubert's Drive, Skegness (Aug. 7).

Merioneth. Panteidal Farm, Aberdovey (Aug. 10).

Salop. The Hayes Farm, Merrington, Bomere Heath, Shrewsbury (Aug. 10).

Staffs. Handsacre Hall Farm, Handsacre, Rugeley (Aug. 8); Strawmoor Poultry Farm, Strawmoor Lane, Oaken, Wolverhampton (Aug 13).

Surrey. 64, Smallholding, Little Woodcote, Wallington (Aug 13).

Yorks. Westkirk Farm, West End, South Cave, Brough (Aug. 9); 205, Dewsbury Road, Wakefield (Aug. 13).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The Conference on Animal Reproduction

Sir,—I was interested to read your report of the Conference on Animal Reproduction recently held at Cambridge, and in particular, your brief reference to the plenary session dealing with *Vibrio* infection. I hope that the papers concerned will be published in full in THE VETERINARY RECORD, and as soon as possible, in view of their importance and interest to the profession.

It would seem from Terpstra's paper that he has identified three types of *Vibrio* organisms from cattle —I presume from vaginal and uterine discharges and/or from the foetal stomach. The first is catalase positive H2S negative and is capable of producing classical *Vibrio* venereal disease with associated herd infertility and abortions; the second is catalase positive, produces a little H2S, and is usually responsible for sporadic abortions; the third is catalase negative and is non-pathogenic.

Rasbech implied that infection other than venereal infection could occur in bulls.

During discussion attention was drawn to the world-wide distribution of this disease and one had the impression that it appeared to be the chief study—if not the only study—of those concerned in the investigation of breeding diseases.

Lawson, in reply to my own questions, gave the figures of his findings in the examination of aborted foetuses sent to Weybridge from various parts of this country. His figures indicated that *Br. abortus* is still by far the most frequently encountered infective agent in abortions.

It is my view that *Br. abortus* is still a very considerable factor in infertility problems—albeit on a much smaller scale than in pre-S.19 vaccination times. Where natural *Br. abortus* infection exists in a herd—particularly where abortions are occurring in non-vaccinated groups of animals—it is not unusual for in-contact vaccinated animals even now to manifest an infertility syndrome of the classical type.

I believe I am correct in saying that the figures for *Brucella* infection showed some slight increase in the year quoted by Lawson. It was surprising, in view of the immense publicity given to the menace to our herds of *Vibrio* infection, to be told that the figures showed a reduction in the number of abortions due to *Vibrio* during the same period. No details were given as to whether the positive *Vibrio* findings had been classified in the three categories mentioned by Terpstra.

The present state of our knowledge appears to be very confused. It is to be hoped that this will be clarified in the near future by more intensive investigation, both in the laboratory and in the field. It seems urgent that there should be greater understanding of the real place of *Vibrio* infection in our breeding problems and of its incidence not only in this country but throughout the world. It would also seem desirable that at least an equal amount of research should be devoted to the investigation of those conditions responsible for the greater number of abortions (at least twice as many as those due to the sum total of all known infective agents usually examined for in laboratory investigations), and that an attempt should be made to ascertain if such abortions are in fact associated with infertility problems in the herds of origin, and what syndrome is manifested.

Yours faithfully,

G. N. GOULD.

12, Landguard Road,
Southampton.

July 30th, 1956.